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Bronchiectasis.—The authors have found in bronchiectasis that the bronchial secretions generally had a high percentage of eosinophils, that is, 10 per cent or more. Search was made for other evidence of allergy, and cutaneous tests were performed. So consistently were high percentages of eosinophils or clinical allergy found in bronchiectatic patients that for over two years all cases of sinusitis and bronchiectasis were studied from an allergic point of view. In fully 90 per cent of cases of bronchiectasis, a definite diagnosis of allergy may be made on the evidence found. The authors feel that most bronchiectasis is caused primarily by basal allergic bronchitis. It is preventable therefore if the basal allergic bronchitis is recognized as such and given intensive treatment before bronchiectasis results. However, even after bronchiectasis has developed, the disease may be successfully combated if treated early. All patients are given treatment by means of desensitization, elimination diets and avoidance of allergens in the environment. The commonest sensitizations are to feathers, pollens, orris root, house dust and wool. Even in the advanced cases definite benefit is usually obtained in reducing the amount of cough and purulent sputum and also in the reduction of nasal obstruction and discharge, when the latter is present. There is abundant evidence in the literature that bronchiectasis and atelectasis are commonly associated. Basal allergic bronchitis with its allergic swelling of the mucosa and outpouring of tenacious secretion combined with bacterial infection and poor drainage seems adequate cause for producing atelectasis. The chain of events is probably first the basal allergic bronchitis, second the atelectasis and third, in a variable length of time, the bronchiectatic dilatation. It is well known that an attack of an acute respiratory infection often initiates prolonged asthma. It seems reasonable to think that the association of bronchiectasis and respiratory infection can be explained on the hypothesis that the respiratory infection is the means of starting basal allergic bronchitis, which eventually results in bronchiectasis.—*Bronchiectasis: A New Conception of Its Etiology which Makes Prevention and Recovery Possible, S. H. Watson & C. S. Kibler, J. A. M. A., July 30, 1938, cxi, 394.*—(*G. L. L.*)

Bronchiectasis.—Bilateral lobectomy in bronchiectasis has been done only rarely. Nine cases have been reported, with one death. The mortality approximates that of unilateral lobectomy, and the procedure is valuable in certain cases. Bronchiectasis, according to Archibald, can be divided into three stages. The first stage is purely bronchitic with no bronchographic changes. In the second stage there is bronchial dilatation, cough and copious sputum which on culture yields mixed organisms but no anaerobes. The third or foetid stage is characterized by foul sputum containing

anaerobes; bronchograms reveal large bronchiectatic cavities. The most important single factor affecting the death rate in lobectomy appears to be the stage at which the operation is carried out. If surgery is carried out before the disease has progressed to the foetid stage, the chances of a fatal empyema are considerably lessened. The two-stage operation is preferable; adhesions lessen the chances of pleural infection, and the adherent lung is utilized by the patient during the operation and is not flapping about in the pleural cavity. With spinal anaesthesia there is apparently less shock, and not so much struggling and coughing as with inhalation methods. The author's two cases of bilateral lobectomy were both young children, with lower lobe disease. In each case preoperative pneumothorax was carried out, and an attempt made to collapse the lung as much as possible. Neither of the cases manifested respiratory embarrassment with the pneumothorax. The pleural surface was then powdered with iodized talc and the air withdrawn to permit the formation of adhesions. If definite adhesions did not form, as determined by intrapleural readings, the lung was again powdered, or a section of rib was removed and the pleural surface painted with 2.5 per cent iodine. In both cases there was an interval of several months between the lobectomies to note any change in signs and symptoms. Both children did very well. Cough and sputum became minimal, clubbing disappeared and general condition improved considerably.—*Bilateral Lobectomy for Bronchiectasis, D. E. Ross, Canad. M. A. J., December, 1938, xxxix, 549.*—(*H. R. N.*)

Lobar Immobilization and Retraction.—Attention is directed to the reaction of immobilization and retraction of a lobe of the lung in the case of a nontuberculous bronchopneumonia. Such a condition has certain specific radiological peculiarities by which it can be recognized. The case of a 13 year old child illustrates this. The patient suffered several attacks of bronchitis and finally the cough persisted. A roentgenogram of the chest showed a soft, light, fresh, exudative pneumonic lesion in the right upper lobe, the lesion being sharply demarcated by the interlobar fissure. Sputum, however, was negative for tubercle bacilli. The clinical course of the illness was stable, there was progressive improvement and a roentgenogram eighteen days later showed complete disappearance of the lesion. A diagnosis of nontuberculous bronchopneumonia was made. Of particular interest in this case was the comparison of the two roentgenograms. The second picture shows the interlobar fissure to be in a new position, visibly lowered and more straight-lined. The internal attachment of the fissure in the hilum is fixed, while its external extremity is quite flat. There is striking evidence of the fissural ascension which evidently took place at the onset of the illness, reducing the size of the lobe of the lung. The authors also noted the slight ascension of the right leaf of the diaphragm. What is the mechanism of this retraction of the lung? The fleeting transient nature of the retraction suggests a functional mechanism, of reflex origin. This localized retraction results in diminished respiratory function placing the diseased area at rest. This is not only a hypothesis but rests on experimental evidence.—*Mise en évidence de la réaction d'immoblisation et de rétraction dans un cas de bronchopneumonie banale (non-tuberculeuse), F. Cardis & A. Gilliard, Arch. méd. chir. de l'app. respir., 1938, xiii, no. 2, 122.*—(*J. E. F.*)

Coccidioides Infection.—*Coccidioides immitis pyogenes*, the infecting fungus agent in coccidioidal infection, goes through two cycles of development. One is the form which occurs when the organism has infected animal tissues, and the other is seen when the organism is grown on culture medium and, presumably, in nature. The parasitic organism is a double contoured spherule, averaging about 30 micra in diameter, and reproducing by endosporulation; the endospores are released into the tissues by rupture of the wall of the mother spherule. When the organism grows outside of the body, it appears as a white mold-like fungus which reproduces by the formation of chlamydospores. The chlamydospores are numerous and light, and easily transported in the air, particularly when associated with dust. Prolonged observation has failed to show any person to person, or animal to person infection. Other methods of transmission, even including insects, have been suggested but none has proved adequate. However, there is unquestionable proof in the literature that inhalation of the chlamydospores of the fungus coccidioides will cause infection in human beings. It has also been shown that the so-called San Joaquin (California) fever, also called "Valley fever" or "desert fever," represents the primary acute coccidioidal infection. The frequency of this disease is shown by the fact that from January 1, 1936 to May 15, 1937 there were 354 recorded cases in the San Joaquin Valley. Valley fever occurs at all seasons and affects both sexes and all ages. At the onset the patient complains of general malaise and an upper respiratory

infection, and a diagnosis of influenza or a bad cold is frequently made. Fever may begin immediately or four or five days later, and varies from 100° to 105°F., when signs of bronchopneumonia appear. There is usually bronchitis, cough and sputum, sometimes blood-streaked. Eight to fifteen days after the onset, tender erythematous nodules appear in the skin, usually on the shins, although they may occur anywhere on the body. These lesions are typically those of erythema nodosum, but sometimes have the appearance of erythema multiforme. These lesions disappear, except for a brownish pigmentation, within four to five days, and then the temperature gradually subsides. There is a moderate leucocytosis up to 15,000, and often an eosinophilia up to 8 per cent. All but one of the 354 patients recovered without complications after an illness of a few weeks; one died of coccidioidal meningitis. The sputum of these patients shows the typical spherules which can also be found in the gastric contents in children. The fungus grows readily on ordinary culture media, but Saboraud medium is preferable for diagnostic purposes. For differential diagnosis of the organism and to establish its virulence, it is necessary to inject the fungus into a guinea pig. If roentgen examination of the chest is made at the time the erythema nodosum appears, densities, often diagnosed as tuberculosis, are seen. Typically there is evidence of enlargement of the hilar nodes accompanied by branching shadows from the hilar regions and scattered areas of parenchymal involvement in either the upper or lower lobes. Signs of pleurisy, pleural effusion or interlobar empyema may be seen at times. In the great majority of cases these pulmonary lesions disappear completely within a few months. The use of coccidioidin is particularly valuable in the diagnosis of Valley fever. This consists of the Berkefeld filtrate of a broth culture of coccidioides which has been incubated from ten to thirty days; sometimes it is modified by grinding a portion of the mycelium of the culture and adding it to the broth before filtration. The method of use is the same as the Mantoux method of testing with tuberculin. The length of time after infection that sensitization occurs in the patient is not known, but in animals infected by inhalation of chlamydospores, positive tests are manifest six or seven days after inoculation. A strongly positive test has been obtained in patients ten years after an episode of Valley fever. The majority of patients personally seen by the authors have been exposed to dust usually in the fields or on the roads. In a few of these acute cases, true coccidioidal granuloma develops. The mortality rate of this later, progressive, secondary phase is approximately 50 per cent. Pathologically, in animals infected by inhalation, in the acute disease, the process appears to be a temporary irritation of the lymphatic system which does not produce tissue destruction or granulomatous changes. When the chlamydospores are injected subcutaneously or into the bloodstream, the reaction is typically granulomatous. Obviously, the well established name coccidioidal granuloma is not applicable to the primary infection produced by inhalation of the infecting organism. The general term coccidioidomycosis is therefore suggested. Individual cases can be classified as primary coccidioidomycosis or progressive (secondary) or granulomatous coccidioidomycosis.—*Coccidioides Infection (Coccidioidomycosis)*, *E. C. Dickson & M. A. Gifford, Arch. Int. Med., November, 1938, lxii, 853.*—(*H. R. N.*)

Spontaneous Pneumothorax.—A review of the literature of idiopathic and tuberculous spontaneous pneumothorax is presented and 24 cases of spontaneous pneumothorax occurring in apparently healthy persons are reported. Three of the 24 were found to have tuberculosis, while no pulmonary disease could be demonstrated in the remainder. The author classifies spontaneous pneumothorax as symptomatic, which occurs as a complication of pulmonary disease, chiefly tuberculosis, and asymptomatic or idiopathic. He is unable to accept infection as the aetiological factor in the latter type, but suggests a modification of the theory recently advanced by Schommer and Erhlich. In brief, his theory is that a congenital pleural defect, or an acquired pleural defect with a congenital anlage may rupture as a result of the normal wear and tear of the pleura during respiration because of its inherent weakness. Such a weakened area in the pleura might even bulge due to tension and form a bleb, and some observers insist that a rupture of a bleb or cyst occurs in all cases.—*Spontaneous Pneumothorax, J. J. Kirshner, Am. J. M. Sc., November, 1938, cxcvi, 704.*—(*G. F. M.*)

Spontaneous Pneumothorax and Bullous Emphysema.—Opportunities of studying spontaneous pneumothorax at necropsy are rare, hence this case diagnosed roentgenographically during life is reported. *Case report:* An air pilot, aged 35, was admitted to the Middlesex (England) Hospital September 16, 1936. For about ten years he had suffered from attacks of dyspnoea and abdominal discomfort and

during the month previous to admission these symptoms had increased and he had lost weight but had continued flying until two days before admission. At that time a consultant found him in severe dyspnoea with heart displaced to the right. Six years ago he had had an haemoptysis. On admission there was bulging of the intercostal spaces on the left side, respiratory movements were much diminished and the trachea displaced to the right, with cardiac pulsation to the right of the sternum. The diagnosis of left spontaneous pneumothorax was confirmed by inserting a needle into the left pleural cavity where an average pressure of +4 cm. of water was found, and 400 cc. of air were aspirated with some relief. Further aspirations were performed September 26 and October 20. A large translucent area was visualized at the left base, bounded by lung tissue, in the X-ray film, and presumed to be a bulla. This showed a fluid level on one occasion. The patient was discharged at his own request October 24 but readmitted ten days later with increasing breathlessness of twenty-four hours duration. He was intensely cyanotic, the left side of the chest bulging and tympanitic. A high intrapleural pressure was found; 2,000 cc. of air were removed and a little later 800 cc. more. New X-ray films showed the left lung collapsed with the bulla still present at the base. November 27 he developed acute tonsillitis and although this subsided he died suddenly December 1. Necropsy showed some adhesions attached to the apex of the left lung and at the base of the lower lobe a large emphysematous bulla which remained inflated, with two smaller ones above it. No bronchial communication was discovered. The pneumothorax, however, was considered to be emphysematous in origin and there was no evidence of tuberculosis (although two small cavities were found in the right upper lobe).—*Spontaneous Pneumothorax Associated with Bullous Emphysema, A. Willcox & A. F. Foster-Carter, Lancet, August 7, 1937, ccxxxiii, 315.—(A. P.)*

Spontaneous Pneumothorax.—Spontaneous pneumothorax has long been associated with pulmonary tuberculosis, but few cases were reported prior to the advent of the X-ray examination and fluoroscopy. Of especial interest is the increasing number of cases of essential or idiopathic spontaneous pneumothorax appearing in the literature. A review of the literature reveals that many employ the term "spontaneous" in the sense that no internal or external cause is apparent, whereas "spontaneous pneumothorax" should include all cases whether primary or secondary to pathological change. "Idiopathic" or "essential" spontaneous pneumothorax is the term applied when there is no evident cause for this condition. The nature of the lung unit or lobule, the pleura and the pleural cavity are described. Attention is called to the numerous divisions of the lobule, the presence of scattered alveoli along the walls of the respiratory bronchioles, the tendency of the lung to collapse. Spontaneous pneumothorax is due to a tear in the visceral pleura and subadjacent tissues. Three main types exist: (*1*) the open type in which air enters and leaves the pleural cavity through the point of rupture during inspiration and expiration; (*2*) the closed type in which the point of rupture quickly closes, the amount of air admitted being variable but usually not large, accompanied as a rule by mild symptoms; and (*3*) the valvular type in which air enters but does not leave the pleural cavity producing mediastinal displacement, some compression of the opposite lung and severe symptoms. Primary spontaneous pneumothorax occurs in apparently healthy persons, is comparatively rare, is found more often in the young and apparently may be induced by only slight exertion. A number of hypotheses have been advanced in explanation of this condition. These include: (*1*) rupture of a pleural tuberculous focus impossible to demonstrate with present methods, (*2*) rupture of an emphysematous vesicle or bulla, (*3*) rupture of a pleural adhesion, and (*4*) the authors' theory of a visceral pleural defect resulting from natural "wear and tear." This latter theory assumes that no matter what the cause of the pleural and lung laceration the location is what determines whether or not pneumothorax will occur. Thus, if the tear is in an area where the communication with bronchi and trachea is fairly direct so that atmospheric pressure may exert sufficient force to drive air into the pleural cavity, pneumothorax will result. However, if the communication is remote, and the pressure exerted is slight as in the intercommunicating alveoli, no pneumothorax will result. The authors believe that in the normal movements of respiration a continuous shedding and regeneration of the pleural mesothelium occurs so that a defect in regeneration, occurring in a favorable area will produce pneumothorax when accompanied by severe respiratory effort. The probability of such a combination of circumstances is remote, hence the infrequency of the phenomenon. Secondary spontaneous pneumothorax is usually associated with tuberculosis but may also occur with other lesions of lung, pleura or mediastinum, and following attempted

pneumothorax. It occurs more frequently in far advanced cases of tuberculosis but may be found in minimal cases arrested for many years. The condition is characterized by a sudden sharp pain in the chest, accompanied by a choking sensation, increasing dyspnoea and sometimes collapse. Often the history and appearance is diagnostic, but the physical findings may not be conclusive unless the collapse is complete producing mediastinal shift. The diagnosis is confirmed by physical examination and X-ray films, for fluoroscopy alone is not sufficient to exclude it. A roentgenogram in expiration and inspiration may be needed in suspicious cases; in all serial films should be taken. The prognosis is good for recovery in the primary type of that secondary to slight pulmonary disease. As a complication of pneumonia the outlook is serious, and when associated with malignancy the prognosis is that of the cause. The open and closed types have a better prognosis than the valvular form in which the symptoms are more serious and complications more prone to occur. When uncomplicated and attended by mild symptoms, the treatment consists of rest and symptomatic medication. Deflation is indicated when dyspnoea is severe. Case reports are presented illustrative of a variety of types of spontaneous pneumothorax. One in this series of cases had three occurrences with complete reëxpansion following each, while in another case the lung failed to reëxpand seven years following collapse.—*Spontaneous Pneumothorax, D. E. Ehrlich & A. Schomer, Radiology, April, 1938, xxx, 471.*—(*G. F. M.*)

Spontaneous Pneumothorax.—Lacerations of the lung at the base of adhesions, with resultant spontaneous pneumothorax, may complicate pneumothorax therapy for pulmonary tuberculosis at any time during the course of treatment and occurs more frequently than is commonly supposed. It usually follows a pneumothorax refill at a time when adhesions are stretched rather suddenly. The danger is greatest in the early stages of an induced pneumothorax and in those cases in which the lung undergoes considerable reëxpansion between refills and requires the introduction of a sufficiently large quantity of air at each filling to maintain the lung in satisfactory collapse. In mild cases the tear may become temporarily sealed and give little or no warning of impending catastrophe, for the spontaneous pneumothorax may recur at any subsequent refill and may become sufficiently alarming to require emergency measures. When adhesions remain attached to the lung and prevent self-closure of the tear, surgical severance of the adhesions responsible (pneumonolysis) is a specific procedure to abolish the complication. The author reports 3 cases in which the operation resulted in immediate recovery after a serious spontaneous pneumothorax developed following a therapeutic refill.—*Spontaneous Pneumothorax Complicating Pneumothorax Therapy: With Recovery after Pneumonolysis: Report of Three Cases, J. W. Cutler, J. A. M. A., July 30, 1938, cxi, 408.*—(*G. L. L.*)

Tension Pneumothorax in Newborn.—A case of unilateral pneumothorax in the newborn, recognized four hours after birth, is reported. The recovery was spontaneous and dramatic after the simple withdrawal of air on two occasions during the first twenty-four hours of life. An unusual feature in this case was the presence of extensive subcutaneous emphysema, which disappeared promptly with the clinical improvement of the infant's condition. Pneumothorax in the newborn results from the trauma at birth; hence the study must extend over the first two or three weeks of life or longer. In any case of neonatal dyspnoea, the chest should be examined both roentgenologically and fluoroscopically. In this way, many occurrences of the condition which are now overlooked would be recognized.—*Tension Pneumothorax in Newborn, Report of a Case with Recovery, H. Strongin, Am. J. Dis. Child., July, 1938, lvi, 110.* (*Author's Summary*).—(*M. B.*)

Primary Cancer of Lung.—At the Cleveland City Hospital, in the period from 1927 to 1937 inclusive, there were 100 cases of primary carcinoma of the lung in which autopsy was performed. There were 35 small cell carcinomata, 40 squamous cell carcinomata, 22 adenocarcinomata and three classified as carcinoma simplex. The term "small cell" is preferable to the terms oat, spindle and round cell, since these merely indicate subvarieties of one general group which is now accepted as arising from the mucosal lining of the bronchial tree. In 78 of the 100 cases the tumor involved a main stem, eparterial or hyparterial bronchus. In 10 cases the tumor was not accurately located in relation to the bronchial tree, and in the remaining 12 the major bronchi were free from tumor and the growth was either in the lung parenchyma (4 cases) or involved a minor branch bronchus (8 cases). The parenchymal carcinomata were circumscribed growths, 5 to 10 cm. in diameter, the origin of which could not be traced to a macroscopically visible bronchus; these were situated in the lung periphery. Small cell

carcinoma: The usual point of origin is at or proximal to the hilum of the lung. Seventy per cent of these tumors arose in a main-stem bronchus. The tumor extended into the lung by direct infiltration, along the bronchi and by way of the peribronchial lymphatics. The pleura was involved in 11 cases, with bloody effusion in eight. Bronchial occlusion was not infrequent and resulted in bronchiectatic abscesses or single large abscess cavities. The tracheobronchial nodes were always extensively involved. In 13 cases there was pericardial involvement and in seven of these the tumor extended into the wall of the auricles. Small cell carcinomata show wide-spread metastases with a tendency to diffuse permeation of the lymphatic system. The presence of carcinoma in superficial lymph nodes was uniformly indicative of extensive involvement of intrathoracic and intraäbdominal nodes. The most frequent sites of metastases were liver, adrenals and pancreas with the brain, skeletal system, kidneys and spleen following in that order. The skin was involved in 14 per cent of the cases; 89 per cent showed some evidence of extrathoracic dissemination. Squamous cell carcinoma: These tumors usually arise distal to the bifurcation of the main-stem bronchus; 70 per cent began in an eparterial or hyparterial bronchus. In 5 cases ribs and thoracic wall were eroded, an observation confined to this type of tumor. Slow bronchial occlusion led to peripheral bronchiectasis (29 cases) and formation of one or more abscess cavities (13 cases). In 8 cases diffuse bronchogenic spread led to consolidation of an entire lobe by tumor. The squamous cell tumor shows no tendency to invade the lymphatic system. Direct extension from the bronchus of origin to adjacent bronchopulmonary nodes frequently occurs. In 65 per cent autopsy revealed no extrathoracic metastases. Metastatic tumor of the opposite lung was found in 5 cases. Adenocarcinoma: In 20 cases, two involved the main-stem bronchus, three originated in a small branch bronchus and the remaining fifteen in eparterial or hyparterial branches. The bronchus of origin was usually occluded by tumor. Pleural infiltration occurred in 50 per cent. Bronchiectatic abscesses were frequent. In 3 cases there was lobar consolidation by tumor with central necrosis and cavity formation. The tracheobronchial nodes were involved in 84 per cent of the cases. These tumors metastasize essentially by way of the blood-stream. The adrenals were most frequently involved (64 per cent). The kidneys, skeletal system, opposite lung, skin and heart were more frequently involved by adenocarcinoma than by small cell carcinoma, while invasion of the liver and pancreas was less frequent and less extensive. Carcinoma simplex: One was a circumscribed growth. The other two involved hyparterial bronchi and showed extensive invasion of the lungs, tracheobronchial nodes, and wide-spread metastases. Seventy-eight biopsy specimens were obtained. Almost half the specimens of small cell tumor were removed from an extrathoracic site; the specimens of squamous cell tumor were almost uniformly obtained from within the chest and in the neighborhood of the primary tumor. Among the small cell tumors, extrathoracic superficial nodes represented 45 per cent of the positive specimens, while there were no positive lymph node biopsy specimens of the squamous cell type. The majority of specimens showing adenocarcinoma came from the lung; lymph nodes were an unimportant source. The average ages were: small cell carcinoma 46 years, squamous cell carcinoma 56 years, and adenocarcinoma 51 years. The ratio of men to women was 9 to 1. Cough, thoracic pain, haemoptysis and dyspnoea were the most frequent symptoms. The complaints in the patients with squamous cell carcinoma were almost uniformly referable to the respiratory system. In the cases of small cell tumor, the frequent formation of a mediastinal mass was responsible for a triad of symptoms not observed in the other types: dyspnoea of the obstructive type, dysphagia and dysphonia. The total duration of symptoms from onset to death averaged six months in the cases of small cell carcinoma, twelve months in the cases of squamous cell carcinoma and eight months in the cases of adenocarcinoma. In the cases of squamous cell tumor the chief source of error in diagnosis was the masking of the tumor by suppuration with resulting picture of pulmonary abscess. Carcinoma was mistaken for tuberculosis in 9 cases. A past history of chronic pulmonary infection was conspicuously absent. Active tuberculosis was present grossly in 2 per cent and healed tuberculosis in 5 per cent. Pulmonary symptoms were absent (4 cases) or slight (8 cases) in 12 patients with tumors showing no relation to a major bronchus. Prognosis is poor in cases of small cell tumor; roentgen therapy does not appear to be efficacious. The squamous cell carcinoma offers the most favorable prognosis for surgical resection. The opportunity afforded for surgical treatment is less favorable for adenocarcinoma than squamous cell carcinoma, owing mainly to the extensive blood-stream dissemination of the latter.—*Pri-*

mary Carcinoma of the Lung, S. Koletsky, Arch. Int. Med., October, 1938, lxii, 636.—(H. R. N.)

Cancer of Lung.—Seventy-three cases of bronchial carcinoma are reviewed. Of these, 65 were proved by histopathological studies. Studies of the material in 62 cases revealed a marked pleomorphism in most with a tendency to show a predominating cell type or group, depending upon the degree of differentiation, the three chief groups being adenocarcinomatous, squamous cell type, and those with anaplastic tendencies. On grouping roentgenographic characteristics, atelectasis, increased markings, and tumor mass were the most frequent findings in the order named. Atelectasis is a common indirect finding in the hilar or central variety and direct visualization of the tumor mass is frequently seen in the peripheral tumors. Single anterior-posterior and lateral films were found to be preferable to the routine stereoscopic films. A unilateral density, in contradistinction to the bilateral shadows as seen in tuberculosis, present in a person over thirty, obligates the examiner to rule out bronchial carcinoma. Neither radical surgery nor irradiation therapy warrant much optimism in the majority of cases.—*Primary Bronchus Carcinoma, W. L. Mattick, Radiology, June, 1938, xxx, 741.—(G. F. M.)*

Cancer of Lung.—The author presents a case report in refutation of the more or less common explanation of the syndrome which has in recent years evoked comment under the designation of "superior pulmonary sulcus tumor." This syndrome is not a clinical or pathological entity, as originally suggested by Pancoast. It can be produced by various tumors invading the tissues near the apex of the lung, most commonly by bronchogenic carcinomata. The term "superior pulmonary sulcus" itself, as indicating the site of origin, has also been criticized and probably should be abandoned. A recent report in the literature described an epithelial malignant tumor of uncertain origin, producing pain in shoulder and arm, Horner's syndrome, erosion of the upper ribs and neighboring vertebrae, atrophy of arm and hand, and a dense roentgen shadow in the region of the pulmonary apex, as being due to a "clinical entity" excluding the lungs as a source, and being anatomically a tumor near the inferior cervical sympathetic ganglion. Fried and others deny such a narrow entity, and the author's case history lends support to this belief and suggests that the most frequent cause of the syndrome is bronchogenic cancer.—*Carcinoma of the Lung Producing Symptoms of "Superior Pulmonary Sulcus Tumor," E. M. Barton, J. A. M. A., August 6, 1938, cxi, 515.—(G. L. L.)*

Cancer of Lung.—In a total of 18,000 patients in whom malignant growths were discovered, 73 had carcinoma of the bronchus, an incidence of 1 in 250. At postmortem examination this lesion was noted with half the frequency of carcinoma of the stomach and was five times more frequent in men than in women. The greatest prevalence was noted between the ages of forty and seventy years. Metastases were most numerous in the bones; the incidence was 38 per cent. Involvement of distant nodes was slightly less frequent, the incidence being 36 per cent. Extension to the adjoining lobes of the lung or to the contralateral lung or regional nodes occurred with approximately the same frequency as distant metastases. Hence, pathologically considered, the prognostic outlook is therapeutically far from promising. Histopathologically, the greatest incidence was found to occur in the squamous cell group, where keratinizing tendencies were noted in 22 and nonkeratinizing in 12. The next greatest incidence occurred in the less differentiated round cell group, in which 14 tumors were classified. Only 7 cases in the series of 73 could be placed anatomically in the peripheral classification. This peripheral group is important from the point of view of operability. A series of 11 cases was designated as an atypical group with a metastatic history of onset. Here pulmonary symptoms are not manifested, and often roentgen evidence is obscure until some time after the patient has sought relief for distant metastatic phenomena, such as enlarged inguinal nodes in one case, dysphagia in others or tumor of the brain as reported by Fried. The most common roentgen characteristics were atelectasis in the hilum or central type and tumor in the peripheral type, which was found in only 11 per cent of the cases in this series. The predominance of a central location of the primary lesion, the tendency to early and distant metastasis and the marked pleomorphic picture all conspire to make the prognosis unsatisfactory by either surgical intervention or irradiation. Earlier recognition and better therapy remain the urgent need.—*Primary Bronchogenic Carcinoma: From the Pathologic and Radiologic Points of View, W. L. Mattick & E. M. Burke, J. A. M. A., December 25, 1937, cix, 2121.—(G. L. L.)*

Apical Pulmonary Tumors.—Since Pancoast in 1924 reported a group of tumors which occurred at the apices of lungs, 60

cases have been reported. These cases have fulfilled Pancoast's criteria for diagnosis: (*1*) roentgen evidence of a neoplasm at the apex of the lung; (*2*) pain referred to the area of distribution of the involved nerves of the brachial plexus; (*3*) atrophy of the small muscles of the hand on the affected side; (*4*) early development of a homolateral Horner's syndrome; and (*5*) roentgen evidence of destruction of the adjacent ribs and vertebrae. However, any neoplasm that involves the brachial plexus in the neck will produce these characteristic signs and symptoms. Two cases are reported, the first of these was a woman, sixty-four years of age, who had a Horner's syndrome, left chest pain and weakness of certain muscles of the left arm. Roentgenograms demonstrated an apical density in the left chest. At postmortem examination there was a large carcinoma involving the entire left upper lobe and infiltrating the brachial plexus. The second case was that of a man with similar symptoms who was found to have a carcinoma which had metastasized to the lung. All of the hitherto completely reported cases have been summarized.—*Tumors of the Pulmonary Apices and Adjacent Regions Involving the Brachial Plexus, H. C. Habein & J. C. Henthorne, Ann. Int. Med., April, 1938, xi, 1806.*—(*A. A. E.*)

Apical Lung Tumors.—The author presents further evidence based on a study of 7 new cases of so-called superior pulmonary sulcus tumors. This evidence, added to that derived from 8 cases earlier reported, forces him to the conclusion that the majority of malignant tumors located in the region of the thoracic inlet are carcinomata of the terminal bronchioles of the lung. The prognosis is very poor. The average duration of life from the onset of symptoms in a series of 12 deceased patients was 13.1 months. Because of the location of these tumors in the thoracic inlet and their mode of extension to the brachial plexus and inferior cervical ganglion, surgery is not indicated. Not one patient in the entire series received any appreciable relief from pain following radiation therapy. Since severe pain about the shoulder radiating down the arm on the affected side is practically the first symptom in all cases, patients thus afflicted should be carefully examined for the other manifestations associated with these tumors in order to rule out a so-called superior pulmonary sulcus tumor. Because of the symptoms, patients having apical lung tumors are generally given a diagnosis of tuberculosis, neuritis or arthritis before a tumor is suspected.—*Apical Lung Tumors: Further Observations with Report of Seven Additional Cases, J. J. Stein, J. A. M. A., October 29, 1938, cxi, 1612.*—(*G. L. L.*)

Road Dust and Lung Tumors.—In a former study the author showed that road dust containing tar caused skin cancer in 70 per cent of surviving mice and increased the incidence of primary lung adenoma. The present study deals, in like manner with the effect of tar-freed road dust and the following conclusions are drawn: road dust minus tar does not cause skin cancer in mice; it stimulates the production of lung tumors but not as extensively as when tar is present. Since the time factor is important, lung tumors do not usually occur in mice during the first year of life. The dust causes no obvious inflammatory reaction in the lung or lymph nodes, except in the tumors themselves. Presence of lung tumors in some of the controls may be due to dust breathed from the contents of their boxes. Tar, either directly or indirectly, stimulates the production of lung tumors in mice and hastens their appearance. Metastases from lung tumors in mice are recorded (in England) for the first time. The majority of lung tumors in dusted mice are malignant. —*The Effects of Road Dust "Freed" from Tar Products upon the Incidence of Primary Lung Tumors of Mice, J. A. Campbell, Brit. J. Exper. Path., June, 1937, xviii, 3.*—(*S. O. P.*)

Clinical Aspects of Silicosis.—Silicosis is a chronic progressive disease whose progress can be followed by the X-ray. As a result of observations carried on over a number of years, it is now possible to measure fairly accurately the length of exposure necessary to produce definite silicosis, demonstrable by X-ray, under a constant set of conditions. In certain districts a five-year period of exposure resulted in clinical symptoms with definite disability, the life expectancy being about ten years from the beginning of exposure, while in other localities men employed from twenty to forty years were still able to put in the regular hour day. The important factor determining the rapidity and severity of the disease is dosage, which is determined by (*1*) the amount of free silica in the dust inhaled, (*2*) the quantity of the dust breathed, (*3*) the length of the exposure and (*4*) whether the latter is continuous or intermittent. These factors obviously vary in different industrial practices. Chief among the individual abnormalities which may account for the

variations found among those exposed to the same conditions are preëxistent pulmonary disease and syphilis. It is more difficult to make a diagnosis of an occasional case in an ordinary clinic or hospital than in industrial practice where the examining physician is familiar with the occupation of his patients and the nature and extent of the hazard. A great deal is known about the symptoms, progress and prognosis of silicosis resulting from moderate or severe exposure, but not so much is known about it in those industries where the exposure is less intense. In the former the picture is clear cut and dramatic, but in the latter the clinical picture may be indefinite and the disease may not be recognized until a resultant tuberculosis calls attention to the pulmonary condition or unless routine examinations are made on apparently normal men known to be exposed. Some cases become evident following pneumonia. Those with "classical silicosis" nearly always die of tuberculosis, and the more intense the silica exposure the sooner tuberculosis develops and the more rapidly it progresses. Statistics show that tuberculosis mortality is higher than the average even in the dusty trades where the hazard is comparatively mild. The chief symptom is dyspnoea, coming on insidiously. In the early stages the patient first notices gradually increasing shortness of breath, a decrease in working ability, especially if employed on piece or contract work, and an increasing tendency to catch cold. He usually has an irritative, unproductive cough but otherwise looks and feels well and may have a tendency to put on weight. Physical examination reveals nothing. The condition may progress to complete disability from dyspnoea, but tuberculosis usually intervenes. When this happens, one of two things may occur: the change may be sudden and dramatic with rapidly progressing symptoms and death in a few months or the tuberculosis infection may progress slowly, sometimes making it difficult to tell whether the patient really has this complication. In advanced silicosis the dyspnoea is extreme. There is no cyanosis. The general appearance is fairly good. The stethoscope still gives little information. Only X-ray films will reveal the pulmonary damage. Many complain of vague gastrointestinal symptoms. Few have haemoptyses without tuberculosis. There are few cardiac complications. The prognosis is bad for the disease tends to progress even after exposure is terminated and sooner or later they succumb to infection. Pneumonia is a dangerous matter for these patients.—*Clinical Aspects of Silicosis, A. J. Lanza, Radiology, March, 1938, xxx, 294.*—(*G. F. M.*)

Aetiology of Silicosis.—The silicosis problem chiefly derives its importance from three factors: first, silica is widely distributed in nature and is widely used in industry; second, once established, there is no known treatment capable of restoring normal lung function, advanced cases progressing in spite of any measures taken; and third, individuals suffering from silicosis frequently succumb to tuberculosis, so that a decrease in the incidence of silicosis should materially reduce the morbidity and mortality due to tuberculosis. Something of the pathology resulting from dust inhalations has been known since the time of Hippocrates, but no clinical or experimental work was done until the latter part of the nineteenth century and early twentieth. It has been shown experimentally and clinically that the pneumonoconiosis characterized by nodular fibrosis is related only to the inhalation of silica-containing dusts. Of the free silicas, SiO_2 compounds, quartz is the most common, occurring as a large constituent of granite, schist and other rocks and as the chief compound of sandstone and quartzite and so forth. Many ores are found in veins that consist almost entirely of quartz. The next most common form of free silica is the amorphous hydrated form known as opal. Other free forms of silica are flint, and, less abundant, tridynite, cristobalite and siliceous glass. The combined forms are known as silicates. It is obvious that many varied occupations will be associated with a silicosis hazard. The first group of such occupations are those concerned in driving tunnels, constructing highways, mining and so forth. Another group includes industries connected with the processing and industrial use of mineral products, the use of sand and gravel for building purposes, the carving of stone, the manufacture and use of abrasives and the processing of various forms of free silica. It has been estimated that nine per cent of all industrial workers are employed in occupations having some silica hazard; thus, about 1,200,000 are potentially exposed to this hazard in the manufacturing and mechanical industries alone. Lanza and Vane estimated that over 500,000 workers in the United States are exposed to silica dust to a serious degree. Experimental work has shown that sharp hard particles other than silica do not produce the characteristic nodular changes in the lung, but that silica in solution or noncrystalline form has a toxic action on tissue resulting in the proliferation of fibroblastic cells. To exert a harmful action the dust must enter the

finer divisions of the lung. Therefore, the particle size is of importance. Very large particles tend to settle out of the air rapidly and very small ones probably are not retained in the lungs but pass out in the expired air. The harmfulness of a given dust is in direct proportion to the number of particles of free silica less than 10 micra in diameter and probably the greatest danger lies in those between 1 and 3 micra. Seventy per cent of the particles found in industrial dust range from .05 to 3.0 micra in diameter. The rapidity with which silicosis will develop depends upon the dosage, which is chiefly determined by the amount of silica present in the air inhaled and the duration of exposure. Intermittent exposure only delays the reaction, but does not prevent the disease. During the rest period some of the irritative action of foreign particles inhaled may subside and the lung free itself of some of the particles. Coal dust and sandstone, for example, are eliminated more readily than materials with a higher percentage of silica. A definite relationship exists between dosage and the severity of the reaction. The presence of inorganic dusts probably has no effect in the reaction to the silica inhaled. The same is probably true of alkalies. A careful detailed occupational history is of importance as an aid in determining the true exposure. From such records it is also possible to determine the average exposure of that individual for comparison with others and complete histories may be very suggestive of the patient's disability, especially when there has been constant change to jobs requiring less effort. A method of analyzing occupational hazards is presented which permits prophesying quite accurately what will be found on physical examination. The rôle of predisposing causes of silicosis has been given much attention. Race, sex, age and climate and temperature are apparently of no importance. Respiratory infection is the chief predisposing and complicating factor. Preëxistent infection is probably the basis of any individual susceptibility. Chronic, local or constitutional infections materially influence the development of silicosis. Infections anywhere in the respiratory tract, whether acute or chronic, are of the utmost importance since they affect the ability of the lung to rid itself of foreign material. Few uncomplicated cases of silicosis are reported, many having an associated infection of the respiratory tract. Recently bronchial spirochetosis has been reported as a frequent complication. The incidence of pulmonary tuberculosis among silicotics is notoriously high. An analysis of the mortality experience of twelve insurance companies over a period of ten years revealed three times as much pulmonary tuberculosis as occurred in an unexposed group; in trades where the hazard is great the rate is about ten times higher. Gardner estimated that about 75 per cent of those who develop silicosis die of tuberculosis. These data make silicosis an important problem from the standpoint of antituberculosis activities. While all the factors considered are of significance, the tendency of silicosis to progress, even though exposure has ended, remains one of the most important aspects of this condition and makes it a serious occupational disease.—*The Etiology of Silicosis, R. R. Sayers & R. R. Jones, Radiology, March, 1938, xxx, 297.*—(*G. F. M.*)

Roentgenological Aspects of Silicosis.—Roentgenology plays an important rôle in the early diagnosis of silicosis. For this reason the responsibility and importance of the roentgenologist is increasing. He should, therefore, be acquainted with the anatomy, histology and physiology of the normal lung and its units. The lung specialist should have studied occupational diseases from every angle and have practical experience in X-ray diagnosis through the observation of the clinical course as seen in serial films of known cases. In addition he should keenly appreciate the importance of the essential technical requirements needed for a correct interpretation of the film, for normal and abnormal densities should be projected on the film so as to give details true to size, shape and outline, without movement of minute lung structures, and with the minimum distortion and exaggeration. To obtain this the author believes that the minimum requirements are a transformer of 400 to 500 milliampere capacity in a tube having a target focal spot no larger than 4.5 mm., with six foot target film distance and an exposure time of one-twentieth of a second. Emphasis is placed on the importance of keeping the time factor constant, preferably at one-twentieth of a second or less to eliminate the factor of movement. Stereoscopic films are preferable to single exposures and serial films are absolutely essential. Lateral views often are helpful and films taken in inspiration and expiration may be of value in certain cases. The minimum requirements discussed are needed in the study of all lung conditions and are strongly recommended. Six X-ray studies of a normal chest taken during the course of one month, a different technique being used for each examination, are presented. Three films appeared not at all alike, and the group demonstrates the danger of attempting to make a diagnosis of early disease with films

obtained under conditions not known to be technically perfect.—*Roentgenologic Aspects of Silicosis, E. C. Ernst, Radiology, March, 1938, xxx, 285.*—(*G. F. M.*)

Bilateral Spontaneous Pneumothorax in Silicosis.—A case of simultaneous bilateral spontaneous pneumothorax complicating silicosis is reported. The patient had worked for five years as a sand-engraver upon glass, and constantly wore a mask. Death occurred from right heart failure six months after development of the pneumothorax. Sputum examination was negative for tubercle bacilli. Necropsy revealed widespread dense fibrosis, with no characteristic tubercle formation. A large calcified apical nodule was found. Tissue inoculation into guinea pigs did not produce tuberculosis, but the author believes tuberculosis was necessary to the development of the silicosis.—*Silicose pulmonaire avec pneumothorax double chez un sableur (Étude anatomo-clinique), C. Roubier, Rev. de la tuberc., November, 1937, iii, 1047.*—(*H. L. I.*)

Experimental Silicosis from Iron Ores.—The inhalation of free crystalline silica can give rise to silicosis but it is not yet known, for certain, to what extent silicates are responsible for this condition. From experiences with silicosis among iron-miners one is obliged to consider the assumption that silicosis can be produced in these workers through the inhalation of granite dust. To study the effect of inhaling various ores, series of rabbits and guinea pigs were exposed to black ore from Kiruna, black ore from Malmberget, black ore from Grängesberg, blood stone ore from Grängesberg, granite in the form of leptite and quartz, respectively. The dust assayed from 25 to 100 mg. per cmm. the first few months, and from 100 to 200 mg. per cmm. the remainder of the time. Concentration of dust in the air varied during the first few months from 10,000 to 30,000 particles per cc., while later it ranged between 100,000 and 300,000 particles per cc. The animals that survived inhaled the dust for fourteen months and twelve days. They were then allowed to live in ordinary air for four months before examination. Dust was delivered continuously night and day. A number of animals that showed pneumonoconiosis or silicosis on autopsy had shown no alteration of the lungs by X-ray at the end of the experiment. No typical silicosis was evident among the animals exposed to the dust of four different Swedish iron ores containing about 5 per cent silica. Relatively marked pneumonoconiosis arose through dust accumulated mainly intercellularly, in greater or lesser cell aggregations, without any signs of real fibrosis. Inhalation of leptite dust produced lung changes similar to typical silicosis, although fibrosis was not so far developed as it was in the control animals with quartz dust. The results suggest that the greatest risk for workers in iron mines is in the granite dust while the danger of silicosis from inhaling ore dust itself should be considered slight.—*Experimental Investigation Concerning the Liability to Silicosis amongst Workmen in Iron Mines, C. Naeslund, J. Indust. Hyg. & Toxicol., June, 1938, xx, 435.*—(*L. F. B.*)

Isolation of Dust from Lungs and Sputum.—A method is described for isolating the dust in pneumonoconiotic lungs by aid of Perhydrol (Merk) containing 30 per cent hydrogen peroxide. The organic matter of the lung is decomposed and transferred into solution. Apart from the effect on carbonates, the attack on liberated dust minerals is weak. Asbestosis bodies sustain the treatment without visible morphological change, but the organic matter contained in its gelatinous envelopes is chemically attacked and more or less destroyed. The method has been found suitable for isolation of iron-ore dust from sputum but requires rather longer time. For mechanical isolation, a fine grating of lung was used with subsequent separation by centrifuging in heavy liquids, among which acetylene tetrabromide diluted with benzene was found particularly useful. The sample of lung is dissected into small pieces and leached in distilled water several times to remove the preservative. It is placed in flasks of about 100 cc. capacity for every 10 g. of substance. Perhydrol is added in small quantities at first, 3 to 5 cc., and then more. The action is later hastened through cautious heating up to 70° to 80°C. The quantity of perhydrol required varies but averages 100 to 125 cc. per 100 g. of wet material.—*Isolation of the Mineral Dust in Lungs and Sputum, N. Sundius & A. Bygdén, J. Indust. Hyg. & Toxicol., May, 1938, xx, 351.*—(*L. F. B.*)

Excretion of Dust.—To study the manner and rate of elimination of dust, the lungs of healthy cats were insufflated with (*a*) bismuth carbonate, (*b*) bismuth subnitrate, and (*c*) powdered lead glass. Cats have a tolerance for anaesthesia, a wide distensible trachea and a slow respiratory rate. After an initial X-ray film, cats were given atropine sulphate intramuscularly before the chloroform-ether anaesthesia. The routine cocainization of the larynx was unnecessary and obtaining tracheal culture swabs, harmful. Through a child's laryngo-

scope a glass tube, attached to a de Vilbiss insufflater, was introduced to within one cm. of the bifurcation and, under fluoroscopy, the insufflation of dust was continued until the desired concentration and penetration were observed. The anaesthesia was then discontinued and the tube removed. Serial roentgenograms were made until the lungs were again clear. The greatest concentration was in the lower portion but in no case was the entire lung tissue affected. Although the three kinds of dust reached the finest bronchioles they did not enter the alveoli. All the cats remained healthy and, without cough, raised and swallowed all the dust in a variable, but short, time. Reinsufflation of the dry dust did not appreciably prolong the excretory time. This indicated that the dust had no injurious effect on the cilia. The soft and amorphous bismuth carbonate particles, 3 to 12 micra in diameter and having a pH of 7.5, were excreted from the lungs within seven hours to three days. The bismuth subnitrate particles, 6 to 12 micra and with pH 3.3, were excreted within the same, or less, time. The angular and hard, powdered lead-glass particles, 20 to 25 micra and with pH 9.1, were swept out of the lungs in seventeen to twenty-four hours. It has been found that this amount of fluctuation in pH cannot be tolerated by isolated preparations, but these living animals were able to render impotent the weak acids and also, but less readily, the alkalies. Except for the lead and barium oxide, this composition was that of silicate found in silicotic lungs. Since the amount of dust insufflated was much more than inhaled dust and yet not one of 60 insufflations caused dust to enter the alveoli, it would seem that healthy lungs could cope with most dusty atmospheres, provided there were adequate rest periods away from the dust. Two cc. of 1:20,000 acetic acid instilled into the lungs was absorbed before the bismuth carbonate was insufflated six to fourteen minutes later and the results were comparable to those given above. Since the lungs have no mechanism other than absorption for keeping fluid in the bronchioles from reaching the alveoli, there is a striking change in the effect produced by instilling the fluid immediately after the dust, or suspending the dust in the fluid. A reversal of the above acetic acid experiment resulted in the dust being carried into the alveoli where roentgenological evidence of it remained for weeks. However, the cats remained healthy and free from cough. It seems possible that in a moist atmosphere particles of suitable dusts might be agglomerated to form a semifluid which would interfere with ciliary action. When 4.5 cc. of saline containing lead glass dust was instilled into the bronchi it reached the alveoli in a few minutes and remained, as was demonstrated by roentgenological and histological examinations. Dilute syrup, instilled into one lung, was found to depress temporarily the ciliary action and to allow the suspended lead glass particles to be carried into the alveoli where they remained for at least three months, although the bronchi and bronchioles were clear the following day. The lung gradually became a shrunken and fibrotic mass with little normal tissue remaining, but the cat's only symptom was malnutrition. The definite mechanism for clearing the alveoli is still obscure. Variation in volume, as with respiration and cough, plays a part as does phagocytosis. Although there was some histological evidence of the phagocytic cells passing through the alveolar walls and up the lymphatics, there was no X-ray evidence of lymph node enlargement. However, the most important protective and excretory mechanism is the excretion of mucus and the propelling motion of the cilia which extend from the larynx to the bronchioles of 0.3 mm. diameter. In the finest bronchioles the ciliated cells are no longer continuous but are interspersed with nonciliated cells. The absence of cilia on the anterior commissura and vocal cords accounts for tuberculous ulcers being more prominent on the posterior commissura. The mucous blanket which must be of the right concentration and amount moves in a clockwise motion and carries out foreign bodies at a remarkable rate. The diameter of the bronchi and bronchioles is known to vary with respiration but this alone would not necessarily cause excretion, but peristalsis would. Peristalsis and cough are probably reserve mechanisms brought into play when the cilia are interfered with as by disease processes or, temporarily, by lipiodol. Dust does not necessarily produce coughing as do irritating fumes. In pneumonoconiosis the cilia usually persist while in bronchitis the ciliated cells may be replaced by goblet cells. The ten cats in which, after some days, the dust reached the alveoli, had bronchitis, bronchopneumonia, or some other morbid condition. Since a healthy bronchial mucous membrane has intact ciliated epithelium and a normal rate of excretion of dust, the technique described here is considered sufficient to give a standard by which one may gauge the delay produced by pathological processes occurring within the lungs.—*Roentgenographic Studies of the Excretion of Dusts from the Lungs*, *A. E.*

Barcley, K. J. Franklin & R. G. Macbeth, Am. J. Roentgenol., May, 1938, xxxix, 673.—(E. M. J.)

Lung Findings in Foundry Workers.—A total of 4,035 foundry workers were studied over a period of four years. A control group from nondusty occupations numbering 2,444 was used. Seven per cent with definite silicotic nodulation was found in the first group, and none in the latter. Of 279 workers with silicosis, 60 (22 per cent) had tuberculosis either active or indeterminate. Diagnoses were made by physical examination, laboratory findings and chest X-ray films. For active cases one of the following must have been present: (*1*) medium moist râles after cough over the lung lesion, (*2*) sputum positive for tubercle bacilli, (*3*) rapid blood sedimentation, or (*4*) extension of shadows on serial films. Serial studies suggest that silicotuberculosis in foundry workers is primarily a reactivation of a previously acquired tuberculosis. Simple silicosis progresses slowly; it does not cause symptoms or incapacity for work unless advanced. The tuberculosis rate among foundry workers is not raised by dust exposure *per se*, but rises only after the silicosis becomes definitely established. The most hazardous foundry occupations are sand-blasting and sand-chipping or grinding of large castings. The author concludes that two procedures will control the foundry hazard: (*1*) elimination at its source of the dust generated by sand-blasting, sand-chipping, sand-grinding and shake-out operations and (*2*) preëmployment and periodic examinations to prevent tuberculous persons from carrying on a dusty trade and to discover new and reactivated cases as they arise.—*Lung Findings in Foundry Workers, a Four Year Survey, O. A. Sander, Am. J. Pub. Health, May, 1938, xxviii, 601.—(S. O. P.)*

Effect of Water on Industrial Dust.—A series of experiments were undertaken to determine the effect on the production of dust of applying water to a block of sandstone. A block of sandstone was struck, about 1,000 times in fifteen minutes, with a mallet and three-fourth inch chisel in a dust chamber. Samples of the circulated dust were taken with the thermol precipitator and in a few cases with a hand pump on ashless filter paper. The stone was worked: dry, dry with water on the surface, wet with a certain amount of water in irregularities of the surface, thoroughly wet throughout its volume, and wet with a half inch of water lying on the surface. The average number of particles per cc., respectively, for these conditions was: 940, 2,400, 1,550, 5,070 and 45. The average median size of the particles, respectively, was: 0.95, 0.42, 0.27 and 0.33 micra. The dust concentration when the stone was worked dry averaged 9.5 mg. per cmm., and when the stone was thoroughly wet throughout its volume it averaged 3.6 mg. Although the mass concentration is about three times greater with the dry stone than with one thoroughly damp throughout its volume, the number concentration is five times higher with the thoroughly wet stone and the size of its particles is considerably smaller. The smaller particles must depend on the presence of water for their formation or for their release into the air. It is suggested that the presence of water or water vapor materially reduces the rate of coagulation of particles into aggregates. Several industrial processes were studied both under dry and wet conditions of drilling. It was found that no particles larger than 5 micra escaped into the air when the rock was drilled wet. It must not be concluded that water is useless in suppressing dust, but it is evident that careful check of dust samples must be taken from time to time to insure that the apparent absence of dust is real.—*The Effect of Water on the Production of Industrial Dust, H. H. Watson, J. Indust. Hyg. & Toxicol., February, 1938, xx, 155.—(L. F. B.)*

Bronchoscopy in Intrathoracic Diseases.—Benign growths are uncommon; however, they may set up early cough, haemoptyses or elevations of temperature. Signs of bronchial obstruction usually develop late. Endobronchial growths are usually readily identified and should be removed with cupped forceps, except angiomata, which are better treated by diathermy. Removal should be followed by aspiration to prevent recurrence. Endobronchial carcinoma is the commonest malignant neoplasm and associated signs of obstruction or infiltration make the diagnosis reasonably certain. Bronchoscopic examination here gives the earliest direct evidence. Specimens can safely be removed for examination except from suspicious areas covered by normal mucosa, when the presence of aneurysm or tuberculosis must be considered. Malignant infiltration is easily recognized by gentle pressure, giving a "woody" feel. Areas of removal should afterward be irradiated. Where removal cannot be done, radon seeds may be implanted. Acute abscess should be aspirated bronchoscopically. Abscesses due to foreign

bodies clear up with their removal. Lavage should be avoided. In chronic abscess other measures are usually necessary. Lipiodol instillation is of value in defining the size and position of the cavity. Bronchiectasis is a condition where bronchoscopy is most useful. The diagnosis is made clinically but the details determined by X-ray after lipiodol, bronchoscopy being most valuable in eliminating foreign bodies or determining the presence of structure or neoplasm. Bronchoscopic aspiration is definitely palliative, also aspiration with lavage, but must be long continued and combined with medical and postural treatment. Lobectomy is now becoming safer and in certain cases is indicated. Asthma is perhaps least amenable to bronchoscopy although some attacks may be aborted by aspiration. Pulmonary tuberculosis when uncomplicated is rarely an indication for bronchoscopy, exception being made in cases where the side of haemoptyses is to be determined or those doubtful cases where uncontaminated bronchial secretions are desired. The mucosa is generally pale, sometimes with granulations.—*Some Uses of Bronchoscopy in Chest Conditions, W. G. Scott-Brown, Brit. J. Tuberc., October, 1937, xxxi, 270.*—(*A. P.*)

Honeycomb Lung.—The honeycomb lung is rare. It has been observed in embryos, newborn, children, adults and in various animals. The cells vary greatly in size and occur singly or in clusters. The walls are frequently of connective tissue, but are often composed of collapsed or emphysematous lung parenchyma. The cells may or may not communicate with a bronchus. The lumen of the cell is lined with cylindrical and frequently with ciliated epithelium. The cyst walls may contain muscle fibres, elastic tissue, cartilage, blood vessels, fat tissue, mucous glands, and even marrow-containing bone. The cysts may be located in almost any part of the lung. The postmortem findings in one case, that of a three year old girl, were typical. The right pleural cavity contained air. The pleura was adherent at the upper part of the right lung. The interlobar pleura was adherent and contained air-filled vesicles. There were a few large, 3 to 4 cm., blebs on the upper and outer lung surface. In the right upper lobe there were many vesicles filled with a gelatinous material. The adjacent parenchyma was atelectatic. There were no blebs in the middle and lower lobes, but the tissue was a yellow gray, or red gray, atelectatic and friable. Beneath the pleura of the left lung were many blebs pinhead to hazel nut in size. The upper and anterior portion of this lung was air containing, and did not crepitate. The rest of the lung was firm. Sectioning revealed many smooth-walled vesicles separated by bands of gray tissue. These were smaller and less numerous in the lower part of the lung. The parenchyma was either normal or emphysematous. In no instance did the blebs or vesicles communicate with bronchi. Histological study showed that the vesicles were either empty or contained in a few instances a red-staining amorphous material. Narrow bundles of fibrous connective tissue and elastic fibres surrounded them. In this layer plasma cells with round and oval nuclei were frequently present. The second case was in a two and one-half year old infant who died with a bilateral patchy pneumonia. During life the Pirquet and Wassermann and Kahn tests had been negative. There were vesicles in both lungs especially peripherally and in the lower lobes. These vesicles were lined with cuboidal cells. Giant cells or multinucleated plasma cells were very prominent. All inflammatory reaction was recent. Both of these cases were probably the result of developmental disturbances involving the lung vesicles. In neither case was there any evidence of chronic inflammation.—*Kasuistische Beiträge zur Kenntnis der Wabenlunge, S. Chodkowska, Virchows Arch., March, 1938, ccci, 746.*—(*C. L. D.*)

Pulmonary Cyst.—A brief discussion of cystic disorders of the lung is presented. A classification of these as (*1*) air containing and (*2*) fluid containing, either form being acquired or congenital in origin, is suggested and attention is called to the fact that "pneumocele" would be a more correct term for the former type. A case is reported of an enormous solitary pulmonary pneumocyst occurring in a healthy adult woman. The patient was clinically asymptomatic and capable of participating in such activities as tennis and swimming despite the fact that the cyst occupied most of the left thoracic cavity resulting in displacement of the heart and mediastinum to the right and flattening of the left diaphragm. Her vital capacity was 27 per cent of the expected normal. The author surmises that there probably is a ball valve action of a bronchus permitting small amounts of air to enter the cyst, and consequently giving it some respiratory function. Films taken during a nine-year interval revealed no significant change. It is believed to be an acquired postinflammatory cyst of emphysematous origin following pertussis and pneumonia in infancy. Only

two other cases are reported having pneumocysts of such size.—*Pulmonary Pneumocyst, G. Cheney & L. H. Garland, Am. J. M. Sc., November, 1938, cxcvi, 699.—(G. F. M.)*

Congenital Cystic Disease.—Because of recent papers questioning the occurrence of congenital cystic disease of the lung, the following report is made. An infant, delivered spontaneously, was blue at birth, improved following respiratory stimulation but again became cyanotic twenty-four hours later. During the following days this became increasingly severe, though oxygen therapy and cardiac stimulation helped some. On the third day bulging of the right chest anteriorly was noted. An X-ray taken at this time revealed atelectasis of the left lung with displacement of the heart and mediastinum to that side, overdistention of the right lung with a number of large irregular air spaces separated by thin partitions of the density of fibrous bands. The child expired on the fifth day. Postmortem examination revealed a normal heart and no free fluid or adhesions in either pleural cavity. There was moderate atelectasis of the left lung. The right upper and middle lobes were largely atelectatic, the air bearing portions showing a moderate degree of emphysema only on microscopical examination. The right lower lobe was greatly enlarged having the appearance of a large thin-walled cavity. Small areas of lung tissue varying from red to dark pink color were present on the edges, crepitant lung being present only on the diaphragmatic surface. The dilated sac-like portion consisted of a single air-filled space, lined with smooth glistening tissue, with fibrous strands partially dividing the cavity. Microscopically the surface was lined with a layer of moderately high bronchial-type epithelium on a definite but variable, connective tissue membrane. Adjacent alveolar spaces opened into the cyst as they do into bronchi. The lung tissue around the cyst wall was normal except for occasional large emphysematous spaces. No macroscopical communication could be found leading to the cyst, but a probe could be passed through the branch of the lower bronchus going to the diaphragmatic portion of the lung.—*Congenital Cystic Disease of the Lung in a New Born Infant, F. A. Ford, Radiology, February, 1938, xxx, 248.—(G. F. M.)*

Sources of Pulmonary Haemorrhage.—Tuberculous patients who bleed may be divided into several categories. Bleeding may originate in ulcerative processes in the larger air passages. Such haemorrhages may be accompanied by fever, often by high swinging temperatures, constant barking cough, small streaks of blood and sometimes larger clots mixed with small amounts of pus and viscid tracheobronchial secretion. The bleeding arises from branches of the bronchial artery. Fatal haemorrhage has been described from this source but is usually not serious enough to demand active treatment. Bronchoscopic treatments to control such bleeding have not been encouraging. Patients bleeding from a cavity are more or less constantly febrile, expectorate amounts of blood, and often continue to bleed once they have started. Two ways to control the haemorrhage are open, one seeking to compress the bleeding cavity and the other a direct attack upon the bleeding vessel itself. Operation is risky but stopping the haemorrhage is of primary importance. If the source of bleeding lies in the pulmonary artery, ligation of it should control the haemorrhage, but if bleeding arises from the bronchial artery, directly or indirectly through dilated anastomoses, then ligation of the pulmonary vessels would be of no benefit and a method that interrupted the bronchial circulation would be needed. The results of seven intrapleural operations to control haemorrhage have been discouraging. Two of the cases recovered. Three were treated by hilar ligature; all died, 2 of circulatory failure and one probably the result of aspiration into the better lung. In one case the pulmonary artery to the diseased lobe was ligated and the patient survived. One cavity was opened and packed; the patient died, probably of progressive disease, two weeks later. One patient with bilateral disease was treated by extensive pneumonolysis and will probably recover. In one, a total pneumonectomy was done and the patient died later of circulatory failure. The fact that bleeding may originate in either the bronchial or pulmonary circulation and that isolated ligation may not be sufficient is of greater interest than the technical features of the operation. Mass ligation of the whole lobar hilum tight enough to occlude vessels, but not the bronchus, is feasible, does not lead to necrosis of the lobe, and seems to check haemorrhage. In the discussion it was stressed that, if a pulmonary neoplasm bleeds, it is more likely to be primary than metastatic.—*Observation on Sources of Pulmonary Hemorrhage and Attempts at Its Control, L. Eloesser, J. Thoracic Surg., August, 1938, vii, 671.—(L. F. B.)*

Atelectasis.—Five cases were considered where obstructive atelectasis, localized in

the medial inferior part of the lung, pre sented a radiographic appearance resembling the paravertebral triangular density of basal exudative mediastinal pleurisy. The distinguishing signs were indications of displacement of adjacent organs to the affected side and the appearance of the triangular, convexly bounded density in oblique or lateral X-ray pictures. In three of the cases the atelectasis was verified by autopsy and in two by lipiodol examination. The clinical symptoms varied with the pathological condition at the root of the bronchial occlusion. The physical signs were dulness and weakened breathing over the base of the lung. The bronchial occlusion was caused by changes in connection with acute bronchopneumonia, bronchial cancer or enlarged lymph nodes in the hilum.—*Lung-Atelectasis with Roentgen Appearance Reminding of Basal Mediastinal Pleurisy, H. Hallander, Acta tuberc. Scandinav., 1938, Fasc. 2-3, xii, 264.*—(*F. B. S.*)

Obstructive Atelectasis.—Although the term atelectasis has been the subject of much criticism and various terms have been suggested in place of it, it would seem proper to use the term atelectasis to the exclusion of all others regardless of the associated pathological phenomena, since the word itself denotes a collapsed state of the lung. Several important factors favoring the development of postoperative atelectasis are bronchial secretion, diminished cough reflex, weakness of thoracic muscles, immobility of the thoracic cage and diaphragm due to pain, and posture. Atelectasis may be patchy or lobular, lobar, or involve an entire lung depending on the bronchus occluded. Because of mechanical obstruction and changes in intrathoracic pressure, transudation of blood elements from the capillaries occurs and liquid accumulates in the lung. Three arbitrary stages may be said to follow bronchial obstruction. There is first a dry or early stage which occurs with the absorption of alveolar gases soon after obstruction occurs. The second or wet stage results from transudation of blood elements and the accumulation of bronchial secretion in the alveoli. The third or fibrotic stage follows a persistent wet stage and denudation of the alveolar epithelium. The radiographic appearance differs in accordance with the stage. In the first, there is moderate density, the result of pulmonary condensation. The second stage resembles, in appearance, a pleural effusion but is differentiated from it by displacement of the heart, trachea and mediastinal structures. In cases of massive atelectasis of long duration the lung may become fibrotic, in which case the X-ray shadow may appear dense and homogeneous as in the second stage.—*Dry and Wet Stages of Obstructive Atelectasis, J. Kaunitz, J. Thoracic Surg., June, 1938, vii, 512.*—(*L. F. B.*)

Bronchial Obstruction.—In a series of experiments several types of foreign bodies were lodged in the bronchial tree for varying periods of time, and the early and late effects noted. The pathological changes noted within the first few days depend principally upon the chemical nature of the foreign body. Vegetable substances containing soluble irritants produced a fulminating reaction in both lungs characterized by oedema and haemorrhage and, in addition, there was evidence of degeneration of the bronchial mucosa and dilatation of the bronchi in the obstructed lobes. There was no evidence of swelling of the peanut or castor bean that was used to produce the obstruction. The slight haemorrhages that occurred were obviously due to disruption of the bronchial wall at the site of lodgment. Inert substances, nail and tooth, produced a less severe type of reaction consisting chiefly of local haemorrhage and ulceration of the bronchial mucosa. In a few instances following the introduction of a tooth, the patchy atelectasis, purulent exudate and petechial haemorrhages were probably on an infectious basis. Late effects appear to depend chiefly upon the completeness of obstruction. If obstruction was complete, there was massive atelectasis distal to the obstruction, suppurative infection and saccular dilatation of the bronchi. Experiments suggest that, at first, obstruction is usually incomplete but may become complete through the accumulation of fibrinous exudate around the foreign body. Non-obstructed bronchi also show cylindrical dilatation and patchy areas of atelectasis and suppuration due to bronchogenic spread of infectious exudate. If obstruction remains incomplete, late changes vary from local ulceration to advanced cylindrical bronchiectasis.—*Bronchial Obstruction Produced by Organic and Inorganic Foreign Bodies, J. Weinberg, J. Thoracic Surg., June, 1938, vii, 488.*—(*L. F. B.*)

Avian Bacilli in Lymph Node of Hodgkin's Disease.—The presence of avian tubercle bacilli in man is very uncommon. L'Esperance reported their presence in lymphogranuloma. The following is likewise a report of the finding of tubercle bacilli of the avian type in Hodgkin's disease. A lymph node from an autopsy of a patient dead of Hodgkin's disease was cultured with the most extreme precautions to avoid

laboratory contamination. About the fifteenth day of incubation acid-fast bacilli were found on the cultures. The microorganisms were inoculated into chickens, rabbits and guinea pigs. All the chickens became markedly positive to avian tuberculin and less strongly positive to bovine tuberculin. They all died with an infection typical of avian tuberculosis. All the rabbits were negative to skin tests with avian tuberculin and slightly positive to bovine tuberculin. The rabbits all died. At their autopsies no lesions could be found, but the visceral organs contained numerous acid-fast bacilli. The guinea pigs all died and were without visceral lesions. One guinea pig had a small nodule at the site of inoculation in the subcutaneous tissue. All the guinea pigs were negative to both avian and bovine tuberculin. Because of these reactions in the laboratory animals the acid-fast microorganisms were classified as tubercle bacilli of the avian type. Six months later the organisms were again inoculated into chickens, rabbits and guinea pigs with results identical to those of the former experiment. From these experiments it was concluded that a tubercle bacillus of the avian type was isolated from a lymph node of a patient suffering from Hodgkin's disease. —*Présence d'un bacille tuberculeux de type aviaire dans un ganglion de lymphogranulomatose maligne, H. Bonnet, S. Thieffry & M. Montefiore, Compt. rend. Soc. de biol., June 18, 1938, cxxviii, 583.*—(*M. T. W.*)

Black Pleural Effusion and Pulmonary Anthracosis.—Black pleural effusions whereby the color is due to a suspension of carbon particles are exceedingly rare. Only one such case, that of Roger in 1903, has been previously recorded. Roger's case was that of a coal miner who expectorated black sputum and had an empyema. Open drainage of the empyema revealed a homogeneous, gelatinous, blackish-blue pus. Examination of the pus showed it to be abacterial and containing many anthracotic particles, some free, others phagocytosed. The authors' case was a 45 year old coal miner whose present illness began with cough and expectoration of mucopurulent sputum which later became distinctly black in color. The patient suffered night-sweats, dyspnoea and loss of weight. In spite of a persistently negative sputum a diagnosis of tuberculosis was made. The roentgenogram showed disseminated micronodular lesions throughout both lungs and an annular shadow in the left lung interpreted incorrectly as a cavity. Therapeutic pneumothorax was started and several months later an effusion appeared in the pneumothorax space, accompanied by pain, fever and malaise. On aspiration the fluid was found to be blackish-blue in color. After several aspirations of black colored fluid, the fluid became dark green in color. Examinations of the fluid showed no acid-fast bacilli, no bacteria on gram stain; there were many leucocytes, neutrophiles 92 per cent, lymphocytes 8 per cent. Culture of the fluid was negative. Sputum examination by concentration and animal inoculation was negative for tubercle bacilli for two years. Careful and extensive chemical examinations of the chest fluid showed the little black particles to be carbon. Regarding the pathogenesis of the black effusion, the authors believe it has been due to a pulmonary perforation even though there was no indication of this happening by changes in intrapleural pressure readings. However, after injecting congo red into the pleural cavity, the patient's sputum showed red streaking. On thoracoscopy there were seen bluish discolorations of the parietal pleura which on minute examination were found to consist of conglomerations of bluish granulations from 3 to 5 mm. in size, some spherical, others triangular. Coffee-colored strings of adhesions were seen. The lung appeared black due to large areas of pigmentation. In the costophrenic sinus was a dark green fluid. A bronchopleural fistula was not demonstrated. A diagnosis of pulmonary anthracosis seems justified in this case on the basis of occupational history, the black expectoration and the roentgenogram of the chest. Concerning the roentgenogram in pulmonary anthracosis the authors note that the lesions are always bilateral, are frequently micronodular and are more abundant in the hilar regions where they tend to form confluent masses. Surrounding each micronodule is an area of emphysema. Hinault and Shaffner distinguish the following forms of anthracosis: (*1*) a nodular form with medium sized nodules in the middle and lower portions of the lung (the micronodular form resembling miliary tuberculosis is rare), (*2*) confluent central opacities, never lobar, (*3*) large bronchopneumonic foci, (*4*) a mixed form.—*Epanchement pleurétique noir compliquant une anthracose pulmonaire, C. B. Udaondo & A. Vadone, Arch méd. de l'app. respir., 1938, xiii, no. 2, 146.*—(*J. E. F.*)

Lung Changes in Arc Welders.—During the past three years a group of 26 electric welders have been under observation. The metal welded was a low-carbon steel (0.1 to 0.2 per cent carbon, 0.1 per cent silica and 99+ per cent iron) with rods of the same composition. Prior to 1932 bare

electrodes were used exclusively. In 5 men of the group, nodular shadows were seen on chest films which simulated silicosis. None of the men were breathing particulate matter to any appreciable degree other than that in the welding fume itself. The men who showed nodulation had done more work inside the tanks, where the fumes were more dense. Five others, who did less work inside the tanks showed only exaggerated trunk shadows. None of the men who had not worked inside the tanks showed any X-ray lung changes, regardless of the length of their employment. It was possible to make postmortem examination of one man who died following an accident. He had been a bare-rod electric welder for eighteen years, with some work inside of tanks from 1922 to 1930. He had no history of pneumonia or bronchitis and had never lost time because of illness. There was a fine black pigment distributed throughout the lungs but there was entire absence of any evidence of scarring or fibrous nodulation. There was no evidence of tuberculosis. The pigment was entirely amorphous and non-crystalline. Chemical analysis yielded 0.096 and 0.089 mg. per cent of silica. The residue after microincineration contained no refractile or crystalline particles that could be identified as quartz, but an intense potassium ferrocyanide reaction was obtained. Microscopically, there was a deposit of pigment uniformly distributed throughout the lung fields and accumulated in the lymphatics. Fibrosis was not apparent nor were the lymphatics occluded. The striking distribution of pigment around blood vessels affords an adequate explanation for the discrete X-ray shadows, and in this case there is sufficient evidence to warrant the conclusion that X-ray appearances of nodules may result from the deposition of iron in the perivascular lymphatics. Unless the welding is done in confined conditions, causing massive concentration of iron in the fumes, deposition does not seem to occur to an appreciable degree. Functional impairment appears to be entirely lacking. The study calls attention to the effect of bare-metal welding, insofar as the deposit of finely divided iron is concerned.—*Chronic Lung Changes in Electric Arc Welders, N. Enzer & O. A. Sander, J. Indust. Hyg. & Toxicol., May, 1938, xx, 333.*—(*L. F. B.*)

Abscess of Mediastinum.—Mediastinal abscess is a rare complication following haemolytic streptococcal infection of the throat. Following acute tonsillitis and pharyngitis a patient, aged 46 years, developed painful and difficult swallowing, pain between the shoulder blades, fever and leucocytosis. Physical and roentgenological examination revealed a mass in the posterior mediastinum. The abscess was drained by entering the posterior mediastinum after paravertebral resection of the right second, third and fourth ribs. A pure culture of haemolytic streptococci was obtained from the pus from the abscess. The patient made an uneventful recovery. Abscesses may occur in the anterior or posterior mediastinum; in either case the diagnosis is made on the basis of the signs and symptoms of infection and characteristic roentgenological findings. The treatment is prompt surgical drainage.—*Abscess of Mediastinum following Acute Tonsillitis, C. S. Keefer, Ann. Int. Med., February, 1938, xi, 1426.*—(*A. A. E.*)

Mediastinitis.—In 60 cases of mediastinitis studied at the Boston City Hospital there were 18 abscesses of the anterior mediastinum (9 acute and 9 chronic), 26 abscesses of the posterior mediastinum (23 acute and 3 chronic), and 16 diffuse phlegmonous infections. These infections may spread along the fascial planes, upward into the neck, down into the retroperitoneal tissues, along the bodies of the vertebrae to the endothoracic fascia, or through the broad ligaments of the lung into the lung substance. Compression of the trachea and oesophagus with actual rupture of the abscess into them occurs. Extension to the pleural cavity has been observed. Roentgen examination reveals triangular shadows superimposed on the cardiac shadow, or rounded or triangular shadows in the superior mediastinum. When the abscess is in the superior mediastinum, the oesophagus is displaced anteriorly. The spine should always be examined for tuberculous or other infections. In 8 acute anterior abscesses the focus of infection was in the neck, larynx, sternum, lymph nodes or lung. Pus in the anterior mediastinum tends to extend to the exterior, pointing at the upper or lower end of the sternum, or at an intercostal space parasternally. Prominent symptoms of acute anterior infection are substernal pain and throbbing pain on movement of the trachea, and swelling and oedema of the neck and thoracic wall with venous dilatation. Tuberculosis is the most common cause of chronic abscess of the anterior mediastinum; it arises from involved lymph nodes, or by extension from the sternum or lung. There were 9 cases of chronic diffuse mediastinitis; 4 were instances of mediastino-pericarditis. Four of these were due to syphilis (with aortic aneurysm in one), 3 to rheumatic fever with mitral stenosis; in 2 the aetiology was obscure. The commonest feature of

this type is compression of the superior vena cava and innominate veins. Recurrent laryngeal paralysis and signs of bronchial stenosis may be associated. In all 4 cases of mediastino-pericarditis there was chronic adhesive pericarditis; the clinical features were those of cardiac insufficiency with congestion, marked by recurrent ascites. The most common causes of acute posterior infection were perforation of the oesophagus by foreign body or tumor, suppuration of lymph nodes and extension of infection from the retropharynx or spine. The common symptoms are pain, usually between the shoulder blades, dysphagia and dyspnoea. Oesophageal compression as seen by barium studies is frequent. Localized collections can be drained by surgery. Recovery in one case occurred after spontaneous rupture of the abscess into the oesophagus. Mediastinal infection secondary to suppuration of lymph nodes was seen following grippe; others have reported this following influenza and pneumonia. Chronic abscess of the posterior mediastinum is almost always due to disease of vertebrae or lymph nodes, most commonly caused by tuberculosis, rarely by actinomycosis or pyogenic infections. The abscess occurs most commonly in the lower cervical and upper dorsal regions. Fever, back pain, dysphagia, cough and gradual wasting are common complaints. Tuberculosis of the spine may be present without roentgen findings. Acute diffuse mediastinitis occurred with pneumonia (14 cases) and peritonitis (2 cases); this is always a serious complication.—*Acute and Chronic Mediastinitis, C. S. Keefer, Arch. Int. Med., July, 1938, lxii, 109.*—(*H. R. N.*)

Mediastinal Cysts.—A case is reported in a seven month old girl who had suffered from attacks of dyspnoea and cyanosis since birth. These symptoms had increased in severity and frequency during the week prior to admission. The child was brought to the hospital desperately ill and lived only eight hours after admission. Clinical findings indicated a mediastinal mass pressing on the left bronchus and so producing atelectasis of the left lung, in which there was a superimposed pneumonia. X-ray films indicated the atelectasis but did not show the mass. Autopsy confirmed the clinical impression and disclosed a congenital ciliated columnar epithelial cyst of the mediastinum. There was bronchopneumonia and early abscess formation of the left lung with a fibropurulent pleuritis on the left. An unexplained hydropneumothorax was present on the right with partial atelectasis of the upper lobe of the right lung. Although small cysts have been discovered accidentally in autopsies of adults who had no symptoms, there are instances reported in adults where such cysts enlarge sufficiently to produce pressure on the respiratory passages. Other case reports indicate that once the cysts become large enough to produce symptoms, there is steady progression of symptoms until death or until operative interference is attempted.—*Congenital Ciliated Columnar Epithelial Cysts of the Mediastinum, Case Report, L. M. Johnston, Am. J. Dis. Child., August, 1938, lvi, 312.*—(*M. B.*)

Diaphragmatic Hernia.—Diaphragmatic hernia is being recognized with increasing frequency. This is almost entirely due to the development of roentgenological diagnosis, which not only confirms the clinician's suspicions but is of aid in determining the size and location of the opening and the abdominal viscera involved. Because of the importance of a history of injury in the diagnosis, prognosis and treatment of diaphragmatic hernia, they are here classified as nontraumatic, either congenital or acquired, and traumatic due to indirect injury or rupture as the result of inflammatory necrosis. The symptoms are complex and sometimes confusing and may resemble those associated with other organic disease of the upper abdomen and thorax. The conditions with which it is most frequently confused are cholecystitis, cholelithiasis, peptic ulcer, hyperacidity, secondary anaemia, cardiac disease, cancer of the cardia, stricture of the oesophagus, appendicitis and intestinal obstruction. In the cases reviewed an average of three previous erroneous diagnoses were made before the correct one was found. The diagnosis is often selfevident at roentgenological examinations, but frequently the case requires critical study as small reducible hernia may escape detection. Among the roentgen signs suggestive of hernia are displacement, tortuosity or angulation of the lower segment of the oesophageal hiatus. Reducible hernia may often be revealed by pressing upward on the stomach. Symptoms are usually progressive, their character depending upon the time in the course of the disease the patient is seen, the rapidity with which it develops, the degree of the fixation of the structures involved and the severity of disturbed function. Para-oesophageal hernia, essentially congenital in origin and having only the stomach involved, is the most common type. The symptoms are more uniform than in any other form of hernia, may begin at any time in life, usually are mild at the

onset and are characterized by epigastric pain transmitted to the back. Belching or vomiting may give relief. Later the attacks occur more frequently and may follow ingestion of any food. The pain is severe, is referred straight back and belching or vomiting may give no relief due to spasm of the diaphragm or reflex cardiospasm. The diaphragmatic spasm commonly produces phrenic pain which is referred to the left shoulder. Dyspnoea, palpitation and tachycardia may occur as the result of pressure on the thoracic structures. Vomiting frequently is severe and of the retention type. Haemorrhage is not common. The symptoms of traumatic hernia are more acute, more severe and more rapid in development; the first often are those of circulatory and respiratory embarrassment. Later haemorrhage may occur as the result of strangulation or incarceration. The only treatment which gives relief of symptoms or prevents complications is surgery. Small hernia of the cardia may be treated palliatively. The repair is usually best accomplished through an abdominal approach using *fascia lata* and interrupted linen sutures. Temporary or permanent phrenic paralysis is a valuable procedure before repair and may be used as a palliative measure in some inoperable cases. The surgical results in 131 cases are reported. Of these, 64 had permanent or temporary phrenic paralysis; in 11 of these the procedure was a palliative measure. Radical repair was performed in 120 cases with 113 recovering. Of these, 110 experienced relief from symptoms. In 3 the hernia and symptoms recurred.—*Clinical and Roentgenologic Manifestations and Surgical Treatment of Diaphragmatic Hernia, with a Review of 131 Cases, S. W. Harrington & B. R. Kirklin, Radiology, February, 1938, xxx, 147.*—(*G. F. M.*)

Acute Suppurative Pleuritis.—The author reports 3 cases of acute suppurative pleuritis, following rupture of lung abscesses that were successfully treated. A large opening was immediately made just lateral to the costal vertebral gutter at the most dependent level of the accumulated fluid. A thick, voluminous dressing was immediately applied, without the insertion of drains, and the patient placed in a semi-recumbent position, with his back against the dressing in order to make the incision air-tight. In a few days, or when pleural and soft tissue edges interfered with continuous drainage several Penrose drains were inserted. No attempt was made to visualize the lung or enlarge the abscess opening or remove devitalized tissue until the patient had recovered from the initial shock.—*Treatment of Acute Suppurative Pleuritis following Rupture of Lung Abscess, F. S. Dolley & J. C. Jones, J. Thoracic Surg., June, 1938, vii, 463.*—(*L. F. B.*)

Radiology of Syphilis of Mediastinum.—The question of the syphilitic mediastinum has not received a satisfactory clinical solution; the nosology not being fixed, diagnosis is always difficult. The difficulty of the problem is illustrated by the following case. A 47 year old patient with painful hepatomegaly and splenomegaly without dropsy and jaundice was observed. The Wassermann test was positive. A year later ascites, oedema of the feet and jaundice developed. A diagnosis of syphilis and cirrhosis of the liver was made. The cardiovascular and respiratory systems were normal. A roentgenogram of the chest revealed the following interesting findings. In both bases of the lungs especially in the right, there are irregular scattered light infiltrations, with irregular borders. The mediastinal shadow is increased in size, being 3 cm. to the right of the spine invading the pulmonary field and forming a vertical line; the aortic shadow is prominent, the upper part projecting itself on the sternoclavicular articulation; above, the cardiac border is poorly delimited and fuses with the basilar infiltrations. An extensive program of antisyphilitic treatment was begun, the ascites decreased but the patient became worse and died eighty days later. During the program of treatment the mediastinal shadow changed significantly. The pulmonary infiltrations disappeared, restoring the outline of the cardiomediastinal borders. Later, the disseminated light infiltrations reappeared along the cardiac and vascular borders and in the bases. These interpretations of the roentgenogram are as disputable as they have been difficult. Syphilis of the mediastinum has been shown in the past as the tertiary sclero-gummatous syphiloma located almost exclusively to the anterior and upper mediastinum engulfing the large vessels at the base of the heart, giving symptoms of compression. This lesion is usually associated with aortic aneurysm. The location and nature of the pulmonary infiltrations in this patient suggests stasis in the venous system. But the antisyphilitic treatment resulted in a manifest regression of the infiltrations giving proof of their syphilitic origin. At autopsy, no sclero-gummatous lesions were found, no aortic aneurysm, no tuberculosis, but there was congestion and pulmonary oedema and discrete fibrous lesions, intrapulmonary

fibrotic mediastinal lymph nodes, syphilitic in origin. It is therefore important to remember that old syphilitics may have specific mediastinal lesions and this may be associated with circulatory stasis.—*Modifications radiologiques du médiastin chez un syphilitique, E. Sergent, M. Fourestier & F. Franchel, Arch. méd. chir. de l'app. respir., 1938, xiii, no. 2, 127.*—(*J. E. F.*)

Behavior of Rat and Human Leprosy in Foreign Hosts.—The inoculation of human lepromata into lower animals results in abortive lesions unsatisfactory for study. However, there occurs in nature a form of leprosy in rats in which the causative organism and histopathology are very similar to the human type. The attempts to infect volunteers by the inoculation of human leprous tissue have usually resulted in failure, whereas murine (rat) leprosy is readily transmitted by ordinary routes of infection from rat to rat. The authors' efforts have been directed toward securing well established progressive infections of rat leprosy in supposedly refractory species in the attempt to secure some guide for the study of human leprosy in the lower animals. In these experiments rats, showing well established lesions of leprosy, were obtained from Dr. E. L. Walker of the University of California. The human leprous material was obtained from a leper with lesions of the nodular type, that is, rather early stage. The authors' summary and conclusions follow: (*1*) Inoculation of murine leprosy into the brain resulted in the development of progressive lesions in monkeys (*M. rhesus*), in rabbits, in white rats and in ordinary white mice, but not in guinea pigs. (*2*) White mice of mixed breeds were also readily infected by injection in the spleen, the liver or the peritoneal cavity, but subcutaneous injection usually resulted only in abortive infection. Mice of some inbred strains frequently developed well marked local lesions on subcutaneous injection and subsequently extensive metastases occurred. (*3*) Murine leprosy has been maintained by serial passage in white mice of mixed breeds for four years. Various routes of inoculation have been used. Massive infections have developed, providing excellent material for experimental purposes. (*4*) The bacillus of rat leprosy produced infection in the murine rodents, white rats and white mice, according to the manner of a microörganism developing in a favorable host, but the circumstances for its multiplication were apparently less favorable in rabbits and monkeys. (*5*) Monkeys (*M. rhesus*) were kept under observation for more than one year after inoculation with human lepromatous material. Lepra bacilli were seen in small numbers in smears of inguinal nodes. Inoculations were made to other monkeys and a similar low grade infection developed during a period of more than one year. Portions of the pia mater of one of these monkeys were implanted intracerebrally in white rats; fifteen months later many acid-fast bacilli were found in the brain of one of these rats. Briefly, human lepra bacilli were maintained in animals for a period of three and three-quarter years but no progressive disease and no active lesions developed.—*The Behavior of Murine and Human Leprosy in Foreign Hosts, A. W. Sellards & H. Pinkerton, Am. J. Path., July, 1938, xiv, 421.*—(*J. S. W.*)

Histology of Murine Leprosy.—Histological studies of local lesions developing at the site of inoculation of murine leprosy in various species of animals (page 421 *ibid.*) were found invaluable in determining whether true infection, in the sense of progressive multiplication of the organisms introduced, had taken place. In animals showing generalized infection a study of the cells in various organs and tissues was made to determine which ones acted as hosts for the lepra bacilli. From this it was observed that, with certain exceptions, lepra bacilli are capable of growing only in mesenchymal cells of a certain type, namely, cells of the reticuloendothelial system. Occasionally epithelial cells, specifically those of the epidermis, testicular tubes and epididymis, become distended with lepra bacilli. This suggests that the relative resistance of epithelial cells to infection may depend on the inability of lepra bacilli to enter them, rather than on intracellular conditions unfavorable to their growth. In taking up lepra bacilli the cells of the reticuloendothelial system are merely carrying out one of their normal functions, the phagocytosis of lipoid structures that are physiologically useless or detrimental to the organism. It is the ability of the ingested bacilli to freely multiply within these cells, instead of being metabolized there, that results in the conversion of these cells into lepra cells and makes possible the development of progressive and eventually fatal disease. As the lesion progresses new lepra cells are formed, not only from the local mesenchymal cells, but also by mitotic division of pre-existing lepra cells, so that the process is in some respects analogous to a neoplasm. The lepra cells do not, however, acquire any of the morphological characteristics of malignant tumor cells. No evidence of

extracellular multiplication of organisms was found. The vacuolated or foamy appearance of the cytoplasm of the lepra cell, which is characteristic of the human lesion, is only rarely seen in murine leprosy. In the progressive lesions produced by *B. lepra murium* in rabbits and monkeys, however, lepra cells are often vacuolated and the acid-fast bacilli within them much less numerous than in rats and mice.—*Histological and Cytological Studies of Murine Leprosy, (with colored plates), H. Pinkerton & A. W. Sellards, Am. J. Path., July, 1938, xiv, 435.*—(*J. S. W.*)

Reactions to Johnin and Tuberculin.—Five patients with cicatrizing enteritis and 5 normal adults were tested intracutaneously with Old Tuberculin and with Johnin made in the same way from Johne's bacillus, but no relation could be established between the enteritis and cutaneous sensitivity to Johnin. In all cases, however, where there were reactions, the larger ones occurred to the Johnin.—*Cutaneous Reactions to Johnin and Tuberculin in Patients with Cicatrizing Enteritis, M. Williams, Proc. Soc. Exper. Biol. & Med., January, 1938, xxxvii, 644.*—(*F. B. S.*)

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